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RECENT STUDIES ON HYDATID DISEASE IN ALASKA

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Before discussing hydatid disease, I should like to make a brief prefatory statement to permit a better understanding of the conditions under which knowledge of this and other diseases has been developed in Alaska.

The approximately 220,000 people making up the population of Alaska at present are separable ethnically and socially into two groups. The majority, about 180,000 people, is comprised essentially of individuals of European descent who live in and around the urban centers of south-central and southeastern Alaska. The health problems of these people differ little from those of residents in most of the other states and need not be considered further here. The second group, numbering about 40,000, or less than 20 per cent of the total, is made up of aboriginal Indians, Eskimos, and Aleuts, most of whom inhabit comparatively remote regions. Many of these aborigines live under conditions that favor the transmission of helminthic diseases.

From 1867, when Alaska was purchased from Russia, until after the end of the Second World War, medical facilities available to the indigenous peoples were at best inadequate. The Alaska Native Service, a branch of the Bureau of Indian Affairs, provided care for many of the more seriously ill, but the program did not include preventive medicine or research. The construction of roads and numerous air fields during and after the war did much to make previously isolated settlements more accessible to medical personnel, and the situation gradually improved.

In 1947, a group of physicians under the auspices of the American Medical Association undertook a survey of medical conditions in Alaska, and the resulting report provided new incentive for some remedial action. Consequently, in 1948, the Congress of the United States authorized the Public Health Service to establish the Arctic Health Research Center. The recruitment of personnel was begun in the autumn of 1948, followed by the establishment of laboratory facilities in Anchorage. The investigation of various problems, including hydatid disease, was begun in the spring of 1949. Another event of importance was the transfer, on July 1, 1955, of responsibility for the medical care of the aboriginal peoples from the Bureau of Indian Affairs to the U.S. Public Health Service. Among other things, this change brought a modern 400-bed hospital in Anchorage under the control of the latter service. The establishment of a collaborative relationship between personnel of the hospital and the Research Center has, during the last few years, given considerable impetus to the investigation of hydatid disease in Alaska.

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* Paper presented at the Symposium on *Echinococcus multilocularis* organized by the « Società Italiana di Parassitologia » and the VII International Congress of Hydatidosis (Roma, 22 September 1960).

TABLE I

NUMBER OF CASES OF CYSTIC AND ALVEOLAR HYDATID DISEASE IN ALASKA (1949-1960)

Date	Cystic	Alveolar
1949	2	–
1950	3	1
1951	1	–
1952	–	–
1953	–	1
1954	–	–
1955	7	–
1956	1	–
1957	3	–
1958	6	3
1959	13	2
1960	4	2

Up to the present time, 41 cases of cystic hydatid disease and 10 cases of alveolar hydatid disease have been treated or otherwise confirmed in Alaskan aborigines. Cases diagnosed since 1949 have been summarized by year in Table I. The comparatively large numbers indicated for the last three years reflect a greater awareness of the problem and an increased effort to diagnose and treat such infections. I should like to summarize briefly the information available on the status of these diseases in Alaska.

CYSTIC HYDATID DISEASE

Echinococcus granulosus has a wide geographic distribution in Alaska, with the wolf and wild ungulates serving as the final and intermediate hosts, respectively, in the natural cycle. Human infections can be expected wherever the people keep dogs as draft animals and engage regularly in the hunting of deer (*Rangifer* and *Alces*).

Adult cestodes have been found in only 17 dogs among 414 thus far autopsied, but the rate of infection varies widely according to location. None was found, for example, in 100 stray dogs from the city of Anchorage, and only 2 dogs were infected among 97 from villages in western Alaska where ungulates generally are unavailable. In villages on the Arctic Coast, from which hunters travel inland to obtain caribou, 7 (10 per cent) of 69 dogs were infected. The highest rate was recorded in villages located in the interior where, of 37 dogs autopsied, 8 (22 per cent) harbored the cestode.

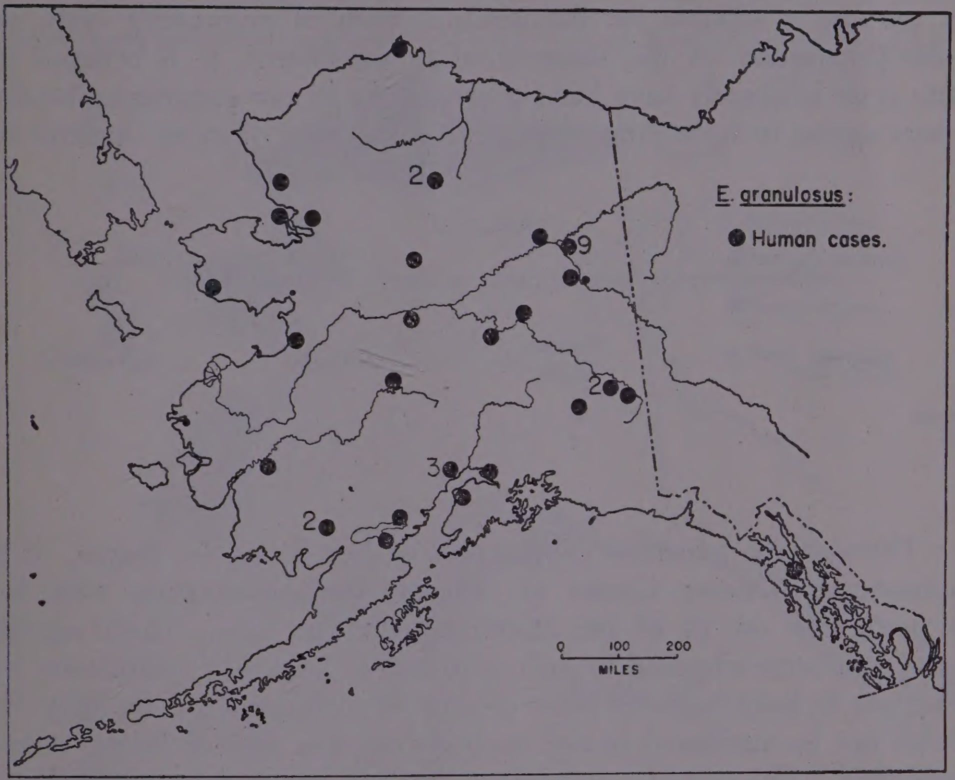

FIG. 1 - Distribution of 40 human cases of cystic hydatid disease in Alaska (1949-1960). Number is indicated if greater than one.

In many villages, particularly those within the forested interior, it can be assumed that dogs having the adult cestode are to be found at all times. Since poor sanitary conditions prevail in most villages, there is a persistent risk of human infection.

The prevalence of infection in the aboriginal population is unknown, for it has not been practicable to undertake an investigation specifically to determine this. The first known Alaskan case, in an Eskimo from the upper Tanana region, was reported by Williams in 1948. From 1949 to the present, 40 additional cases have been found; the locations from which these originated are shown in Fig. 1. Of these, 30 were pulmonary and 10 were hepatic in location; thus far, none has been found in other parts of the body.

Nearly all cases have been found incidental to routine roentgenological examinations for pulmonary tuberculosis. The technique as applied for the latter purpose is suitable for the demonstration of pulmonary cysts, but, because penetration of the tissue is often insufficient, it is believed that hepatic cysts frequently have been overlooked. In our experience, serological tests appear to have little value in the diagnosis of cystic hydatid disease. Through the generous cooperation of Dr. Irving G. Kagan, of the Communicable Disease Center in Atlanta, hemagglutination tests have been performed on 18 of the aforementioned 40 cases. Titres regarded as significant were obtained in only 3 patients. In one, a pulmonary cyst was known to have ruptured prior to surgical treatment, and the high titre probably can be attributed to the antibody response elicited by the escaped hydatid fluid. The second patient, an 8-year-old girl, had an asymptomatic pulmonary cyst that was later removed, while in the third case the diagnosis of hepatic infection was based upon roentgenological findings.

TABLE II.

Disposition of human cases of cystic hydatid disease

Location	Number Treated	Untreated	Mortality
Pulmonary	30	0	0
Hepatic	1	9	0

The disposition of the 40 cases is shown in Table II. Since it usually is not possible radiologically to distinguish an *Echinococcus* cyst from a tuberculoma, all such pulmonary lesions have been routinely removed. The problem of diagnosis is complicated by the fact that tuberculosis has been a very common disease among the aborigines, and the proportion having a positive tuberculin reaction is quite high. In two patients, lesions tentatively diagnosed as cysts were found after surgical removal to be tuberculomas. It has not usually been considered advisable to attempt surgical treatment of hepatic infections.

ALVEOLAR HYDATID DISEASE

Echinococcus multilocularis evidently is restricted in its distribution to the tundra biome of northern and western Alaska, including certain islands in the Bering Sea (Fig. 2). The natural cycle involves foxes (*Alopex* and *Vulpes*) and microtine rodents, but dogs and other canids may harbor the adult cestode. It is believed that human infections are acquired primarily from sledge dogs. The cestodes have been found in 5 of 89 dogs autopsied on St. Lawrence Island, but no adequate survey has been made in the endemic areas of mainland Alaska.

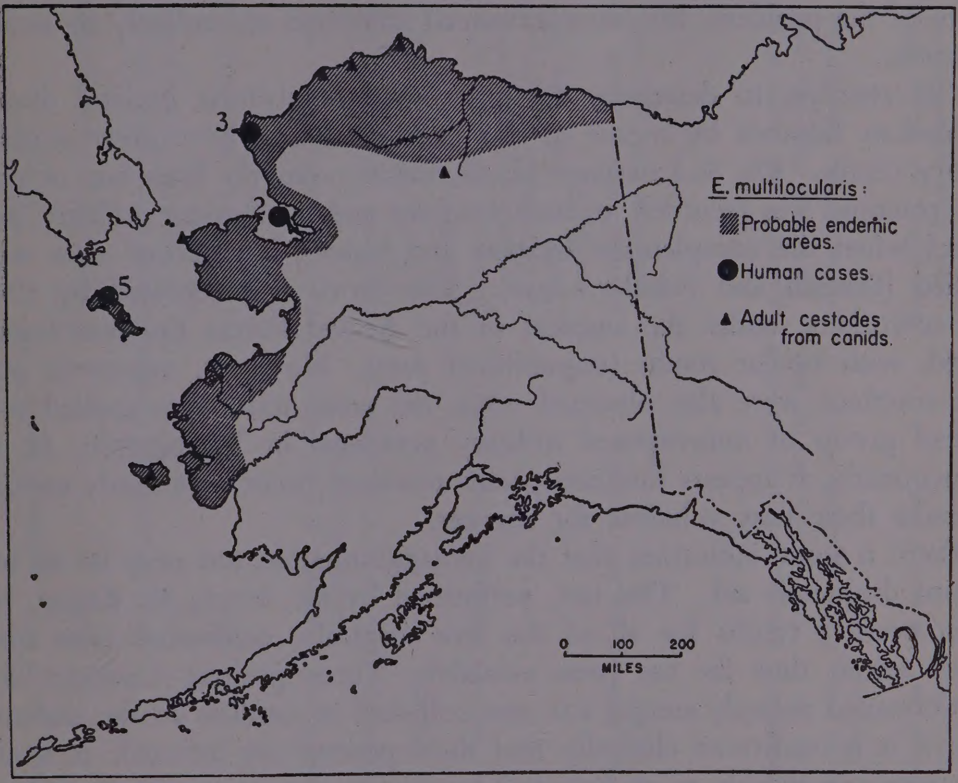

FIG. 2 - Probable distribution of *Echinococcus multilocularis* in Alaska. Designated also are villages in which human infections have been diagnosed, and locations at which adult cestodes have been found in canine animals.

Ten cases of alveolar hydatid disease, all in Eskimos, are known thus far; all of these patients were hospitalized for the treatment of disorders stemming directly from the infections. The findings in these cases are summarized in Table III.

A reliable technique is not known at the present time by which alveolar hydatid disease can be diagnosed in the early stages, but even far advanced infections are frequently overlooked. In Switzerland, for example, Schmid (1958) noted that only 18 of 67 cases confirmed at autopsy had been correctly diagnosed. Several years ago typical hepatic lesions were incised and drained of necrotic material in at least three Eskimos from St. Lawrence Island; the diagnosis was later confirmed in two of these when further surgical treatment was necessary, but the third died before additional information was obtained. At the present time, with medical personnel generally aware of the problem, the more advanced infections are unlikely to escape attention.

Past attempts to determine the prevalence of alveolar hydatid disease in Alaskan Eskimos by means of immunological tests have given unsatisfactory results. On St. Lawrence Island, a comparatively high rate of positive reactions was recorded in both Eskimos and in transient military personnel when the complement fixation and Casoni intradermal tests were applied (Rausch and Schiller, 1956). This work was repeated by Otto and coworkers, under the auspices of the Armed Forces Epidemiological Board, with similar results (unpublished data). However, numerous positive reactions were also obtained when the same tests were applied to a control group of non-exposed military personnel in Washington, D. C. Consequently, it appears that nonspecific reactions occur commonly enough to make these tests valueless for surveys.

There is some indication that the hemagglutination test may be an important diagnostic aid. This test, performed by Dr. Irving G. Kagan, has given positive results for all of the five surgically confirmed cases from which serum thus far has been available. Three positive reactions have been obtained recently among 101 sera collected at random in the endemic area; if it is confirmed clinically that these patients are infected, it would then seem practicable to undertake a survey to determine the prevalence of infection.

Roentgenological findings have been important in the diagnosis of several cases, since the hepatic lesions often contain calcified areas that have a characteristic appearance. Radiographs of such lesions have been figured and described by Friedrich (1940).

Since the infection is pathologically similar to a malignant hepatic tumor, treatment of alveolar hydatid disease is successful only when the lesion can be removed surgically; the prognosis in advanced cases is usually very poor. Thus far, four Alaskan cases have been treated with evident success (Table III). The results of the radical hemihepatectomies that were perform-

TABLE III - DATA ON 10 HUMAN CASES OF ALVEOLAR HYDATID DISEASE

Case	Age	Sex	Clinical findings	Location of lesion	Treatment	Outcome
1	55	M	Mass in liver.	Liver.	Lesion incised and drained *ca.* 1950	Persistent abdominal fistula; died Dec., 1954.
2	58	F	Mass in liver.	Liver; recurrence in abdominal wall.	Nodule excised March, 1947; lesion removed from abdominal wall July, 1959.	Recovered; probable residual infection.
3	65	F	Mass in liver.	Liver.	Lesion incised and drained March, 1951; lesion removed *in toto* May, 1953.	Recovered.
4	29	M	Epileptiform convulsions; signs of tumor in brain.	Postfrontal area of brain; presumably also in liver.	Lesion in brain excised Nov., 1950.	Died July, 1951, in home village; no autopsy.
5	55	M	Hepatomegaly; positive hemagglutination test. Later showed grand mal-type seizures.	Right lobe of liver; brain.	Laparotomy with biopsy of liver Oct., 1957; staged marsupialization of lesion in spring-summer, 1958.	Died August, 1958; autopsy disclosed severe infection right hepatic lobe and 4 cm. lesion in cerebellum.
6	62	F	Hepatomegaly; icterus; positive hemagglutination test.	Mainly in left lobe of liver.	Laparotomy with biopsy of liver March 3, 1958; laparotomy and exploration of lesion on March 21, 1958; lesion later marsupialized.	Died Nov., 1958, in home village: no autopsy.
7	73	M	Not diagnosed *pre mortem*; hepatomegaly and icterus noted.	Right lobe of liver.	Untreated; died as result of hemorrhage from gastric ulcer.	Died May, 1958; 6 x 9 cm. lesion in right lobe of liver found at autopsy; also metastatic nodule 3 x 1 cm. in lower lobe of left lung. (Brain not examined at autopsy).
8	53	F	Radiological findings characteristic; positive hemagglutination test.	Retroperitoneal mass at level of liver; possibly also in liver.	Laparotomy with biopsy of retroperitoneal lesion Feb., 1957.	Inoperable; patient still living. In good health in April, 1960.
9	46	F	Hepatomegaly; radiological findings characteristic; positive hemagglutination test.	Right lobe of liver.	Hemihepatectomy, right lobe, May 1960.	Recovered.
10	22	F	Hepatomegaly; characteristic radiological findings; icterus, pruritis; positive hemagglutination test.	Left lobe of liver.	Laparotomy and biopsy Feb., 1960; hemihepatectomy, left lobe, March, 1960.	Recovered; larval tissue left in right lobe; recurrence of disease probable.

ed in two instances suggest that this may be the best surgical procedure if the lesion is confined to a single lobe. Multiple lesions or lesions involving major blood vessels and bile ducts are inoperable, and the importance of early diagnosis and treatment is obvious.

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